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FAY L. SMIGEL, Widow of
EDWARD SMIGEL,

Claimant

v.

DIRECTOR, OWCP

Respondent

Date Issued: January 11, 2001

Case No 2000-BLA-715

DECISION AND ORDER GRANTING BENEFITS

This proceeding arises from a claim for benefits under the Black Lung Benefits Act, 30 U.S.C. § 901 *et seq.* (the "Act"). The Act and implementing regulations, 20 C.F.R. parts 410, 718, 725 and 727 (the "Regulations"), provide compensation and other benefits to: (1) living coal miners who are totally disabled due to pneumoconiosis and their dependents; (2) surviving dependents of coal miners whose death was due to pneumoconiosis; and (3) surviving dependents of coal miners who were totally disabled due to pneumoconiosis at the time of their death (for claims filed prior to January 1, 1982). The Act and Regulations define pneumoconiosis, commonly known as black lung disease, as a chronic dust disease of the lungs and its sequelae, including respiratory and pulmonary impairments, arising out of coal mine employment. 30 U.S.C. § 902(b); *see* 20 C.F.R. § 718.201. In this case, the Claimant, Fay L. Smigel, widow of Edward T. Smigel, alleges that she is the surviving dependent of Mr. Smigel, whose death was due to pneumoconiosis. The Director of the Office of Workers' Compensation Programs ("OWCP") has not designated any employer as the responsible operator in this case and is therefore the only Respondent.

I conducted a hearing on this claim on October 4, 2000, in Pittsburgh, Pennsylvania. Both parties were afforded a full opportunity to present evidence and argument, as provided in the Rules of Practice and Procedure, 29 C.F.R. Part 18. At the hearing, Claimant's Exhibits 1-2 and Director's Exhibits 1-32 were admitted into evidence without objection. Tr. at 6-7.[1] The record was held open after the hearing to allow the parties to submit additional evidence and argument. I hereby admit the following additional exhibits which have been submitted timely by the parties: CX 3, DX 33, DX 34 and DX 35.

In reaching my decision, I have reviewed and considered the entire record, including all exhibits, the testimony at hearing and the arguments of the parties.

PROCEDURAL HISTORY

Edward T. Smigel filed a claim for black lung benefits on June 28, 1973. DX 27-1. The claim was denied by the OWCP on January 14, 1980, because the evidence did not show that he was totally disabled by pneumoconiosis. DX 27-30 and 27-26. Mr. Smigel did not appeal that decision further.

On January 12, 1984, Mr. Smigel filed a second ("duplicate") claim. That claim was denied by Administrative Law Judge ("ALJ") Edward J. Murty, Jr., in a Decision and Order issued on October 31, 1988. ALJ Murty found that Mr. Smigel was a coal miner for over ten years, that x-ray evidence was positive for pneumoconiosis, and that Mr. Smigel was entitled to the presumption that his pneumoconiosis was the result of his coal mine employment. ALJ Murty went on to find, however, that Mr. Smigel had no impairment as the result of his pneumoconiosis, and, therefore, was not entitled to benefits under the Act. DX 28-46.

Mr. Smigel died on November 9, 1999. DX 4. Thereafter, Mrs. Smigel filed her survivor's claim. Because her claim was filed after January 1, 1982, and Mr. Smigel was not found entitled to benefits as a result of his claim filed prior to January 1, 1982, Mrs. Smigel's claim is governed by the regulations found at 20 C.F.R. Part 718. *See Neeley v. Director, OWCP*, 11 B.L.R. 1-85 (1988).

ISSUE

The only issue in this claim is whether the miner's death was due to pneumoconiosis. DX 29; Tr. at 5. Because the Director is not contesting any other issues, only the evidence necessary to a determination of this issue will be addressed in detail in the decision.

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[1]The following abbreviations are used for reference within this opinion: DX, Director's Exhibits; CX, Claimant's Exhibits; Tr., Hearing Transcript; Dep., Deposition.

APPLICABLE STANDARD

A surviving spouse is entitled to benefits if the miner died due to pneumoconiosis. 20 C.F.R. §§ 725.212(a)(3) and 718.205. In claims filed after January 1, 1982, death will be considered to be due to pneumoconiosis if (1) competent medical evidence establishes that the miner's death was due to pneumoconiosis; (2) pneumoconiosis was a substantially contributing cause or factor leading to the miner's death or the death was caused by complications of pneumoconiosis; or (3) the presumption set forth at 20 C.F.R. § 718.304 applies, i.e., an irrebuttable presumption that death was due to pneumoconiosis where there is medical evidence of complicated pneumoconiosis; but not if (4) the miner's death was caused by a traumatic injury or the principal cause of death was a medical condition not related to pneumoconiosis, unless the evidence establishes that pneumoconiosis was a substantially contributing cause of death. 20 C.F.R. § 718.205(c). The Third Circuit, in which this claim arises, has held that any condition that hastens the miner's death is a substantially contributing cause of death for purposes of this regulation. *Lukosevicz v. Director, OWCP*, 888 F.2d 1001 (3rd Cir. 1989).

FINDINGS OF FACT AND CONCLUSIONS OF LAW

Factual Background and the Claimant's Testimony

The parties stipulated that Mr. Smigel worked as a coal miner for at least ten years. Tr. at 5. He last worked in a mine in Ehrenfeld, Pennsylvania, in 1953. DX 27-7. From 1953 to 1984, he was employed as a welder by Bethlehem Steel. DX 27-7; Tr. at 12. He also welded truck bodies at W. J. Thiel & Sons from 1961 to 1963. Tr. at 23-24. Mr. and Mrs. Smigel were married on June 26, 1978, and lived together until Mr. Smigel died. Tr. at 11. Because they married after Mr. Smigel left the mines, and after he had stopped smoking tobacco, Mrs. Smigel could not testify to the conditions under which he worked in the mines or his smoking habits. Tr. at 11-12. Nor could she say whether he was exposed to fumes or dust as a welder, because he did not discuss his work with her. Tr. at 24. He retired because he was becoming sickly with gall bladder problems, and he had enough years in to retire. Tr. at 12-13. He had his gall bladder removed at Lee Hospital in 1984. Mr. Smigel had also had pericarditis in 1979, but recovered well from that. Tr. at 13.

Mrs. Smigel testified that she first noticed Mr. Smigel having problems with shortness of breath in 1985, after he retired from Bethlehem Steel. His problems started slowly at first, and then got worse. She noticed him having problems with the stairs first. It eventually took him three tries to get up the eleven steps from the basement to the first floor because he had to stop every three or four steps. It could take him 15 minutes, and eventually, 20 minutes, to cover the 40 feet from the garage at the back of the yard. He gave up hunting and fishing almost immediately after he retired because of his problems with shortness of breath. Tr. at 15-16.

In 1997, Mr. Smigel had diverticulosis and a prostate resection. Mr. Smigel had a heart attack while visiting his brother at a nursing home on November 5, 1999. He was taken to the hospital, and died four days later, never having awakened after surgery, on November 9, 1999. Before his heart

attack, his breathing was very poor. He was going downhill every day. Tr. at 13-14, 17.

Medical Evidence

The medical evidence as to Mr. Smigel's state of health in 1988, when his last claim for black lung benefits was denied, is summarized in ALJ Murty's Decision and Order. X-ray evidence was positive for pneumoconiosis and the Director conceded its existence. Mr. Smigel had also been diagnosed with ischemic heart disease. Pulmonary function tests and arterial blood gas studies were normal, and there was no evidence of cor pulmonale with right sided congestive heart failure. ALJ Murty found no impairment as a result of Mr. Smigel's pneumoconiosis. In view of the availability of an autopsy report, there is no reason to address further the medical evidence up to 1988. There is a gap in the records in the file after 1988; the next available records cover the period from 1997 until Mr. Smigel's death in November 1999.

Treatment Records from 1997 to 1999

DX 6 contains Mr. Smigel's records from Lee Hospital from 1997 up to and including his last illness and death. In late 1997, he was hospitalized with a diverticular hemorrhage and bladder outlet obstruction which led to a transurethral resection of prostate. He was also diagnosed with early dementia at that time. In November 1998, after experiencing chest pain and shortness of breath while walking, a cardiolite treadmill evaluation revealed stress-induced ischemia. After he complained of difficulty walking due to unsteadiness on his feet, physical examination and nerve conduction and EMG studies in September 1999 disclosed that he had peripheral neuropathy of unknown etiology. Other than an occasional reference to a history of chronic obstructive pulmonary disease, there were no references to his pulmonary condition in the treatment records other than in connection with his cardiac symptoms. Mr. Smigel was under the primary care of Dr. Thomas Ellenberger until he was transferred to the care of Dr. Philip Rice on November 5, 1999, after his last hospital admission.

Mr. Smigel was admitted to the hospital due to an acute myocardial infarction with pulmonary edema on November 4, 1999. Cardiac catheterization revealed triple vessel disease, which resulted in bypass grafting. After surgery he developed acidosis, renal dysfunction and ischemic insult to the right parietal area, with sudden onset of seizure activity. He never regained consciousness. He did not respond to treatment, and according to the final summary dictated by Dr. Rice,

> He eventually expired from sudden onset of ventricular arrhythmia and electromechanical dissociation with failure to pace despite all chemical intervention and died of severe obstructive coronary disease, status post myocardial infarction with closure of the left anterior descending, and postoperative ventricular failure.

Autopsy Report

Will Farthing, M.D., a pathologist, conducted an autopsy limited to the chest cavity on November 9, 1999. The final anatomic diagnoses were acute myocardial infarction, 4-vessel coronary artery disease, status post coronary artery bypass graft surgery, and cardiomegaly with left ventricular hypertrophy; simple coal workers' pneumoconiosis; and bilateral hemorrhagic pleural effusions. In the portion of the report entitled "Clinical-Pathological Correlation," Dr. Farthing stated:

> The patient presented on 11/4/99 with evidence of acute myocardial infarction. After cardiac catheterization, semi-emergent coronary bypass graft surgery was performed. Post-operatively, the patient suffered sequelae of circulatory shock with hypoxic encephalopathy and acute tubular necrosis of kidneys. . . . Autopsy limited to the chest cavity revealed the above changes, confirming pre-mortem clinical diagnoses.

With respect to the diagnosis of pneumoconiosis, Dr. Farthing described "[s]everal . . . 0.5-2.0 cm. subcarinal anthracotic lymph nodes" and moderate bilateral anthracosis, more prominent in the upper lobes. Microscopic examination of the lungs showed

> some areas of normal architecture and other areas with alveolar clubbing consistent with mild emphysematous change. Many areas of dense anthracosis are noted, with anthracotic black pigmented macrophages. Many of these dense anthracotic areas are perivascular, and some are subpleural. The anthracotic pigment is within macrophages, and some surrounding brown pigment is also noted. Iron stains of these areas show dense accumulation of irons admixed with the anthrocotic pigment. Polarization reveals rare 1-2 mm polarizable particles suggestive of silica. These anthracotic perivascular changes appear to be more prominent in the upper lobes than the lower lobes. No asbestos bodies are identified on iron stain. A small focus of bronchopneumonia is noted in a random section of right upper lobe, with intra-alveolar acute inflamation and fibrin. . . .

DX 5.

Death Certificate

The death certificate appears as DX 4. It lists the cause of death as cardiopulmonary failure due to (or as a consequence of) myocardial infarction. It is signed by Philip Rice, M.D., who had treated Mr. Smigel while he was in the hospital.

Medical Opinions

Dr. Thomas Ellenberger

Thomas R. Ellenberger, Jr., M.D., Mr. Smigel's primary care physician, submitted a letter in

which he stated:

> The above-named patient was followed by me in my office as a general internist over about two years. . .
>
> In November of 1999, the patient was hospitalized on an "emergent basis" because of chest pain consistent with an acute myocardial infarction. He was shortly thereafter found to have "severe triple vessel coronary disease," and underwent an emergency attempt at coronary artery bypass.
>
> The patient's course was complicated by a cerebral infarction and respiratory insufficiency.
>
> It is apparent that pulmonary fibrosis based on coal worker's pneumoconiosis would have been a factor in his problem of respiratory insufficiency necessitating mechanical ventilator support.
>
> The factor of pneumoconiosis has only become more clear related bo a post mortem examination completed shortly after his death . . .

DX 24. Dr. Ellenberger's qualifications and office treatment records are not in the file.

Dr. John Michos

John Michos, M.D. reviewed Mr. Smigel's records and provided an opinion regarding the cause of death on behalf of the Director. Dr. Michos is board certified in internal medicine and pulmonary medicine, and as a medical examiner. DX 32. Dr. Michos opined that Mr. Smigel had simple pneumoconiosis with no clinically significant pulmonary impairment, as demonstrated by normal arterial blood gas studies performed in1984 and 1988, and normal pulmonary function studies performed in 1985 and 1988. Dr. Michos stated that the cause of death was complications encountered after cardiac surgery. He concluded:

> As to the issue of this gentleman's cause of death, it is quite clear it was from complications encountered after his bypass cardiac surgery. Mr. Smigel suffered from severe lactic acidosis, anoxic encephalopathy, and seizures, presumably from emboli during bypass surgery and use of balloon pump. In no way did simple CWP lead to his severe coronary artery disease or respiratory insufficiency, thus leading me to believe that the same outcome would have occurred had the miner never stepped in the mines.

DX 31.

Dr. Joshua Perper

Joshua Perper, M.D., reviewed Mr. Smigel's records, examined autopsy slides and provided

an opinion regarding the cause of death on behalf of the Claimant. Dr. Perper is board certified in anatomical pathology and forensic pathology. CX 2. His microscopic diagnoses were myocardial infarction, slight simple coal workers' pneumoconiosis, mild to moderate focal centrilobular emphysema, and focal bronchopneumonia. Based on scientific literature, he opined that Mr. Smigel's emphysema was attributable to a combination of factors including smoking and coal dust exposure. He stated that the primary cause of death was myocardial infarction, and that pneumoconiosis was a substantial contributory cause directly and through the associated emphysema "that caused hypoxemia that either triggered or aggravated the myocardial ischemia . . . or aggravated the . . . ischemic heart condition following the advent of the myocardial infarction." He went on to state:

> The mechanism of death of the coal worker's pneumoconiosis and associated emphysema was both direct hypoxemia, and complicating acute bronchopneumonia that resulted in a terminal cardiac arrhythmia, on the background of cardiomegaly and moderate arteriosclerotic coronary heart disease.

CX 1.

Dr. Perper was deposed on November 14, 2000. CX 3. He has a medical degree and a law degree, has practiced in pathology and forensic pathology for 35 years, and is a consultant in black lung cases for the Department of Labor. Dep. at 3-5. He was not consulting for the Department of Labor in this case. Dep. at 6. Dr. Perper confirmed that he had reviewed Mr. Smigel's medical records and prepared a report. Dep. at 8-9. He agreed that 15 years of coal mine work is enough exposure to contract pneumoconiosis. He said that Mr. Smigel had a significant smoking history, but the fact that he had stopped smoking in 1970 meant that there was no worsening of his condition based on smoking. Dep. at 11-12. Chest x-rays were positive for pneumoconiosis, "with a profusion of 1/1." Dep. at 12. He agreed from the medical history that Mr. Smigel complained of shortness of breath on exertion beginning in 1979, and that later blood gas studies showed evidence of resting hypoxemia. Dep. at 12-13. He noted that Mr. Smigel was also being treated for arteriosclerotic heart disease, which limits the amount of blood to the heart. Dep. at 13-14. This combination of problems reduced both the flow of oxygen in the blood and the concentration of oxygen in the blood. Dep. at 14. In 1999, Mr. Smigel had a heart attack and was eventually put on a respirator and given additional oxygen before he died. Dep. at 14-15. Diagnoses after autopsy included coal workers' pneumoconiosis and centrilobular emphysema. Dep. at 15. Dr. Perper reviewed the autopsy lung slides and sections which showed scarring, fibrosis, deposition of pigment (fibro-anthracosis and silica crystals), centrilobular emphysema, nodules up to 9 mm, and clusters of macrophages with anthracotic pigment in the alveoli. Dep. at 16. The heart showed fibrosis and scarring and areas of myocardial infarction, with coronary arteries occluded up to 80%. Dep. at 16-17. Dr. Perper testified that the combination of arteriosclerosis and hypoxemia due to pulmonary disease made myocardial infarction "much more likely to happen." Dep. at 17.

With regard to the cause of Mr. Smigel's death, Dr. Perper stated:

> . . . the major cause of death was myocardial infarction and . . . coal workers' pneumoconiosis was a substantial contributory cause of death, both directly and indirectly [due] to hypoxemia and the fact that . . . chronic obstructive pulmonary disease and emphysema which in substantial part was due to coal workers' pneumoconiosis . . . [may] trigger, precipitate or cause a myocardial infarction and a fatal arrhythmia.

Dep. at 18-19. He went on to state that centrilobular emphysema is caused by both smoking and coal workers' pneumoconiosis, which have a synergistic effect, increasing the risk. Furthermore,

> . . . in this particular case, this individual stopped smoking almost 30 years prior to this death, and as I said before, chronic emphysema doesn't become worse with cessation of smoking, while it becomes worse with coal workers' pneumoconiosis because basically the exposure to intrapulmonary silica continues because the silica . . . cannot be . . . excreted from the lungs and stays there . . .

Dep. at 19. He also stated that had Mr. Smigel not been suffering from emphysema and pneumoconiosis, he would have had a better chance of surviving a myocardial infarction if he developed one. Dep. at 20.

On cross examination, Dr. Perper stated that inhalation of welding fumes is not a cause of silicosis, which results from exposure to silica (silicon oxide) contained in rock or stone, and that exposure to silica separately from coal would cause a distinctive lesion without coal pigmentation not present in Mr. Smigel's lungs. Dep. at 21-23. Dr. Perper stated that normal pulmonary function and blood gas studies performed in 1988 would not be significant in judging Mr. Smigel's condition in 1999. Dep. at 24-27. In response to a question whether coronary disease could produce shortness of breath, Dr. Perper testified that Mr. Smigel was experiencing shortness of breath "when there was no heart disease documented clinically or radiologically or by EKG." Dep. at 30. He said that lactic acidosis can occur as a result of myocardial infarction, and as a result of a respiratory contribution to myocardial infarction. Dep. at 31. He said that Mr. Smigel's renal failure was a result of his general poor condition and not primary kidney disease. Dep. at 33.

Dr. Leon Cander

Leon Cander, M.D., reviewed Mr. Smigel's records on behalf of the Director. Dr. Cander is board-certified in internal medicine. DX 35. In his initial report dated October 5, 2000, Dr. Cander noted Mr. Smigel's history of cigarette smoking, coal mine employment and chest x-rays read as negative and positive for pneumoconiosis. He also noted Mr. Smigel's history of welding, and stated that inhalation of welding fumes may cause silicosis. He also reviewed the results of pulmonary function tests and arterial blood gas studies from the 1970's and 1980's, all of which were normal, and noted that blood gas studies from Mr. Smigel's final hospitalization were also normal, later noting that normal or supernormal results would be expected because he was on a ventilator. Dr. Cander reviewed records of hospitalizations and medical examinations between 1973 and 1998, and emphasized that

there was no objective evidence of chronic pulmonary disease or treatment during that period. Based on his review of the records of treatment during Mr. Smigel's final illness, Dr. Cander found it significant that there was no mention of any pulmonary problem or evidence of cor pulmonale. Rather, his treatment was for a critical cardiac condition with coronary artery disease and left ventricular dysfunction caused by myocardial infarction. In Dr. Cander's view, the autopsy documented the cause of death as the complication of severe obstructive coronary disease in its "most lethal form." He went on to state,

> . . . There is no evidence of ventilatory insufficiency or hypoxemia which could have adversely affected cardiac efficiency or cardiac rhythm. There is no question, based upon the report of two independent pathologists that Mr. Smigel had evidence of centriacinar emphysema. What neither pathologist has addressed is the fact that centriacinar emphysema which affects the most distal and tiny ramifications of the airway may not have any significant effect on airway resistance or alveolar gas exchange and arterial PO2. It is well known that the correlation between pathologic evidence and pulmonary function is poor and that it is not possible to predict pulmonary function from slides of lung tissue. In Mr. Smigel's case there was unequivocal evidence of normal ventilatory function and normal arterial oxygen tension both at rest and during exercise during the period September 12, 1973 to his death on November 9, 1999. The centriacinar emphysema could not possible [sic] have hastened or caused his death.

DX 33.

Dr. Cander later reviewed Dr. Perper's deposition and prepared a second report dated December 21, 2000. DX 34. Dr. Cander disagreed with several aspects of Dr. Perper's testimony. Dr. Cander stated that contrary to Dr. Perper's statement, lung function in smokers who have stopped smoking "continues to decrease at a rate greater than that caused by aging alone." Dr. Cander characterized all of the available blood gas studies performed on Mr. Smigel as normal, "excluding the presence of pulmonary insufficiency," and stated that there was no hypoxemia present in any of the studies. Although he agreed that hypoxemia can cause arrhythmia and angina, he observed that complete coronary artery occlusions are caused by blood clot formation. He found no evidence in the record to indicate that Mr. Smigel had chronic obstructive lung disease. He provided an article published by the U.S. Department of Health and Human Services in support of his contention that welding can cause silicosis. He challenged Dr. Perper's statement that silica, as opposed to carbon dust, is the cause of coal workers' pneumoconiosis. He said that "all patients with clinically significant chronic obstructive lung disease reveal decreased pulmonary function test values." He disagreed with Dr. Perper's statement that electrocardiograms did not show coronary heart disease, citing the July 13, 1998 EKG showing "classic changes of ischemia, documenting the presence of clinically significant coronary heart disease." Finally, he said that the lactic acidosis noted in Mr. Smigel's records was cause by decreased cardiac output cause by proven congestive heart failure, with "no evidence of any abnormality of lung function causally related to the lactic acidosis or heart failure."

Whether Pneumoconiosis Was a Substantially Contributing Cause to Mr. Smigel's Death

The record is conclusive in this case that Mr. Smigel had simple pneumoconiosis as a result of his coal mine employment. ALJ Murty so found; the Director has conceded it; and the autopsy has confirmed it. Of the four alternative grounds for entitlement of a surviving spouse to benefits in the Regulations, this case turns on whether pneumoconiosis was a substantially contributing cause to Mr. Smigel's death. According to the *Lukosevicz* case cited above, Mrs. Smigel's burden is to establish that pneumoconiosis "hastened" Mr. Smigel's death. In *Lukosevicz*, the deceased miner died of pancreatic cancer. The death certificate and autopsy report also listed pulmonary emphysema as a contributing cause of death, and the doctor who performed the autopsy stated that anthracosis revealed upon microscopic examination "shortened Mr. Lukosevicz' life, albeit briefly and was therefore contributory to his death. However, his primary cause of death was pancreatic cancer unrelated to his anthracosis." 888 F.2d at 1003 (emphasis added). The Third Circuit concluded that the Benefits Review Board "erred as a matter of law in failing to interpret the regulatory language 'substantially contributing cause' to encompass the situation where pneumoconiosis actually hastened the miner's death" and remanded the case for immediate payment of benefits. 888 F.2d at 1006. Applying this standard to the case at hand, I find that Mrs. Smigel is entitled to benefits because I find that pneumoconiosis hastened Mr. Smigel's death.

In this case, the death certificate identifies the cause of death as cardiopulmonary failure due to myocardial infarction. Two doctors, Ellenberger and Perper, have stated that pneumoconiosis was a contributing factor, while two, Michos and Cander, hold the opposite opinion. All of the physicians who provided medical opinions did so based on adequate underlying documentation. All provided at least some rationale in support of their conclusions. Thus I consider all of these medical opinions to represent documented and reasoned medical opinions. Dr. Ellenberger and Dr. Michos did not elaborate on the reasons for their opinions. Dr. Ellenberger's qualifications are not contained in the file. Dr. Michos, on the other hand, never examined Mr. Smigel and could not have known his level of functioning before his final hospitalization. Both Dr. Perper and Dr. Cander possess excellent qualifications, and both offered extensive explanations of their opinions. I cannot accept everything in either's reports or testimony uncritically, however, as both appear to have made some statements more consistent with advocacy than with objectivity. On balance, however, I find that Dr. Ellenberger's and Dr. Perper's opinion are entitled to greater weight because they were more consistent with the evidence as a whole.

None of the doctors challenge the diagnosis of pneumoconiosis. In some respects, Dr. Cander's criticisms of Dr. Perper's testimony at his deposition appear to rely on aspects of his testimony taken out of context. For example, Dr. Cander states that Dr. Perper believes that silica, rather than coal dust, is the primary cause of pneumoconiosis. Dr. Perper's report makes clear, however, that he was discussing silica as an element of coal dust, albeit an element which is the "major fibrogenic (scar producing) element in coal dust." But Dr. Cander, too, goes on to state that silica exposure causes fibrosis; thus it appears that the two are more in agreement than disagreement on the results of exposure to silica, whether the source was coal mining or welding. Even if Dr. Perper was in

error whether welding could have been a source of silica exposure, the autopsy report characterizes the occurrence of silica as "rare," but refers to "many areas of dense anthracosis." Thus the autopsy findings support the view that coal dust exposure, rather than silica exposure, was the predominant finding. Although Dr. Cander's report suggests that he would give greater weight than Dr. Perper to Mr. Smigel's smoking in the development of emphysema, he does not dispute Dr. Perper's fundamental assertion that coal dust exposure contributed to the condition of Mr. Smigel's lungs.

The primary area of disagreement between Dr. Cander and Dr. Perper appears to be in the area of the degree of impairment caused by the condition of Mr. Smigel's lungs. The problem for both is that there were no pulmonary function studies performed after 1988, and no blood gas studies after 1988 except when Mr. Smigel was on a ventilator, which Dr. Cander acknowledged would have elevated oxygenation beyond what he would have experienced breathing on his own. Dr. Cander's statement that there was "unequivocal evidence of normal ventilatory function and normal arterial oxygen tension" until Mr. Smigel's death obscures the 11-year gap in testing and the use of a ventilator during his final illness. The progressive nature of pneumoconiosis is a fundamental underpinning of the Act and Regulations. The available treatment records do not show what symptoms or treatment, if any, Mr. Smigel had for his breathing after 1988. Mrs. Smigel credibly testified that Mr. Smigel experienced progressive problems with his breathing. The letter from Dr. Ellenberger suggests that he may not have attributed symptoms reported to him to pneumoconiosis until after Mr. Smigel's death. The medical records do refer to a history of chronic obstructive pulmonary disease, as well as pneumoconiosis and emphysema, however, contrary to Dr. Cander's assertion that there is no such evidence. Absent well reasoned and documented medical opinions and objective findings, I could not find that Mr. Smigel was disabled by pneumoconiosis. On the other hand, Dr. Cander's report does not establish that the absence of a disabling impairment is the same as no impairment of lung function. Furthermore, Dr. Cander's response to Dr. Perper's deposition testimony does not negate Dr. Perper's point that chronic obstructive pulmonary disease and emphysema can precipitate a myocardial infarction, and as well as lessen the possibility of surviving one. Dr. Ellenberger's opinion also carries great weight because he was Mr. Smigel's treating physician. The opinions of Dr. Ellenberger and Dr. Perper that pneumoconiosis resulted in some impairment of lung function and contributed to respiratory insufficiency after Mr. Smigel's bypass surgery, support the conclusion that it hastened his death. That is all that is required under the Act and Regulations as they have been interpreted in the Third Circuit where this case arises.

FINDINGS AND CONCLUSIONS REGARDING ENTITLEMENT TO BENEFITS

The Claimant has met her burden to establish that pneumoconiosis was a substantially contributing cause to Edward Smigel's death and is therefore entitled to benefits under the Act.

ATTORNEY'S FEES

The Regulations address attorney's fees at 20 C.F.R. §§ 725.362-367. Claimant's attorney has not yet filed an application for attorney's fees. Claimant's attorney is hereby allowed thirty days

(30) days to file an application for fees. A service sheet showing that service has been made upon all parties, including the Claimant, must accompany the application. The parties have ten days following service of the application within which to file any objections. The Act prohibits the charging of a fee in the absence of an approved application.

ORDER

The claim for benefits under the Act filed by Fay L. Smigel on November 15, 1999, is hereby GRANTED.

ALICE M. CRAFT
Administrative Law Judge

AMC:sjn

NOTICE OF APPEAL RIGHTS: Pursuant to 20 C.F.R. § 725.481, any party dissatisfied with this Order may appeal to the Benefits Review Board within 30 days from the date of this Order by filing a Notice of Appeal with the Benefits Review Board, 200 Constitution Ave., NW, Washington, D.C. 20210. A copy of a notice of appeal must also be served on Donald Shire the Associate Solicitor for Black Lung Benefits. His address is Frances Perkins Building, Room N2605, 200 Constitution Ave., NW., Washington, D.C. 20210